What is claimed is:

1. A method for treating or reducing the advancement, severity or effects of neoplasia comprising the step of administering a therapeutically effective amount of a LT-α/ß heteromeric complex and a pharmaceutically acceptable carrier.

2. The method according to claim 1, wherein the LT-α/ß heteromeric complex has a LT-α1/ß2 stoichiometry.

3. The method according to claim 1, wherein the LT-α/ß heteromeric complex is a soluble LT-α/ß heteromeric complex.

4. The method according to any one of claims 1-3, wherein the LT-α subunit is selected from the group consisting of lymphotoxin-α, native human or animal lymphotoxin-α, recombinant lymphotoxin-α, soluble lymphotoxin-α, secreted lymphotoxin-α, lymphotoxin-α muteins, or lymphotoxin-α-active fragments of any of the above.

5. The method according to any one of claims 1-3, wherein the LT-ß subunit is selected from the group consisting of lymphotoxin-ß, native human or animal lymphotoxin-ß, recombinant lymphotoxin-ß, soluble lymphotoxin-ß, secreted lymphotoxin-ß, lymphotoxin-ß muteins, or lymphotoxin-ß-active fragments of any of the above.

6. The method according to claim 3, wherein the LT-ß subunit is cleaved between amino acids 44 and 88 and the N-terminal portion replaced with a type I leader sequence.

SUB A3

7. The method according to claim 6, wherein the type I leader sequence is the vascular cell adhesion molecule 1 (VCAM-1) leader sequence.

8. The method according to any one of claims 1-3, wherein the LT-α/ß heteromeric complex is administered in the presence of a therapeutically effective amount of at least one LT-ß-R activating agent.

SUB A4

9. The method according to claim 8, wherein one LT-ß-R activating agent comprises a therapeutically effective amount of IFN-γ.

10. The method according to claim 9, wherein a second LT-ß-R activating agent comprises a therapeutically effective amount of an anti-LT-ß-R antibody.

11. The method according to claim 10, wherein the anti-LT-ß-R antibody is a monoclonal antibody.

12. The method according to claim 11, wherein the anti-LT-ß-R monoclonal antibody is selected from the group consisting of anti-LT-ß-R mAb BKA11, CDH10, BCG6 and BHA10.

13. A method for treating or reducing the advancement, severity or effects of neoplasia comprising the step of administering a therapeutically effective amount of at least two LT-ß-R activating agents and a pharmaceutically acceptable carrier.

SUB A5

14. The method according to claim 13, wherein at least one LT-ß-R activating agent comprises an anti-LT-ß-R antibody.

15. The method according to claim 14, wherein the anti-LT-ß-R antibody is CBE11.

16. The method according to claim 13, wherein the LT-ß-R activating agents comprise at least two anti-LT-ß-R monoclonal antibodies which are directed against non-overlapping epitopes of LT-ß-R.

17. The method according to claim 16, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1 and BDA8, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of BCG6, BHA10, BKA11, CDH10 and CBE11.

18. The method according to claim 16, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of BCG6 and BHA10, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1, BDA8, BKA11, CDH10 and CBE11.

19. The method according to claim 16, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of BKA11 and CDH10, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1 and BDA8, BCG6, BHA10, and CBE11.

20. The method according to claim 16, wherein one anti-LT-ß-R monoclonal antibody is CBE11, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1, BDA8, BCG6, BHA10, BKA11, CDH10 and CBE11.

21. The method according to claim 16, wherein the anti-LT-ß-R monoclonal antibodies are CBE11 and BHA10.

22. The method according to claim 16, wherein the anti-LT-ß-R monoclonal antibodies are CBE11 and CDH10.

23. The method according to claim 16, wherein the anti-LT-ß-R monoclonal antibodies are AGH1 and CDH10.

24. The method according to any one of claims 13-23, wherein one LT-ß-R activating agent is IFN-γ.

25. A method for treating or reducing the advancement, severity or effects of neoplasia comprising the step of administering a therapeutically effective amount of cross-linked anti-LT-ß-R antibodies as a first LT-ß-R activating agent in the presence of a second LT-ß-R activating agent and a pharmaceutically acceptable carrier.

26. The method according to claim 25, wherein the cross-linked anti-LT-ß-R antibodies are non-covalently immobilized on a surface.

27. The method according to claim 25, wherein the cross-linked anti-LT-ß-R antibodies are covalently immobilized on a surface.

28. The method according to claim 25, wherein the cross-linked anti-LT-ß-R antibodies are non-covalently aggregated in solution by means of an anti-LT-ß-R antibody cross-linking agent.

29. The method according to claim 28, wherein the anti-LT-ß-R antibody cross-linking agent comprises a secondary antibody directed against the anti-LT-ß-R antibody.

30. The method according to claim 28, wherein the anti-LT-ß-R antibody cross-linking agent comprises an Fc receptor which binds to the anti-LT-ß-R antibody.

31. The method according to claim 25, wherein the cross-linked anti-LT-ß-R antibodies are covalently aggregated in solution by means of a chemical anti-LT-ß-R antibody cross-linking agent.

32. The method according to any one of claims 25-31, wherein the second LT-ß-R activating agent comprises IFN-γ.

33. A method for treating or reducing the advancement, severity or effects of neoplasia comprising the step of administering a therapeutically effective amount of at least one LT-ß-R activating agent and a pharmaceutically acceptable carrier.

34. The method according to claim 33, wherein at least one LT-ß-R activating agent comprises an anti-LT-ß-R antibody.

35. The method according to claim 34, wherein the anti-LT-ß-R antibody is CBE11.

36. A method for selecting a LT-ß-R activating agent which acts in the presence of LT-α/ß heteromeric complexes comprising the steps of:

a) culturing tumor cells in the presence of LT-α/ß heteromeric complexes, an effective amount of a first LT-ß-R activating agent and a second putative LT-ß-R activating agent; and

b) determining whether the second putative LT-ß-R activating agent increases the anti-tumor activity

of the LT-α/ß heteromeric complex in the presence of the first LT-ß-R activating agent.

37. The method according to claim 36, wherein the first LT-ß-R activating agent is IFN-γ.

SUB A4

38. The method according to claim 36, wherein the LT-α/ß heteromeric complex has a LT-α1/ß2 stoichiometry.

39. A pharmaceutical composition comprising a therapeutically effective amount of a LT-α/ß heteromeric complex and a pharmaceutically acceptable carrier.

40. The pharmaceutical composition according to claim 39, wherein the LT-α/ß heteromeric complex has a LT-α1/ß2 stoichiometry.

41. The pharmaceutical composition according to claim 39, wherein the LT-α/ß heteromeric complex is soluble.

42. The pharmaceutical composition according to any one of claims 39-41, further comprising a therapeutically effective amount of at least one LT-ß-R activating agent.

43. The pharmaceutical composition according to claim 42, wherein one LT-ß-R activating agent is IFN-γ.

44. The pharmaceutical composition according to claim 42, wherein one LT-ß-R activating agent is an anti-LT-ß-R antibody.

45. The pharmaceutical composition according to claim 44, wherein the anti-LT-ß-R antibody is a monoclonal antibody.

46. The pharmaceutical composition according to claim 45, wherein the anti-LT-ß-R monoclonal antibody is selected from the group consisting of anti-LT-ß-R mAb BKA11, CDH10, BCG6, and BHA10.

47. A pharmaceutical composition comprising a therapeutically effective amount of at least two LT-ß-R activating agents without exogenous LT-α/ß heteromeric complex, and a pharmaceutically acceptable carrier.

SUB A7

48. The pharmaceutical composition according to claim 47, wherein at least one LT-ß-R activating agent comprises an anti-LT-ß-R antibody.

49. The pharmaceutical composition according to claim 48, wherein the anti-LT-ß-R antibody is a monoclonal antibody.

50. The pharmaceutical composition according to claim 49, wherein the anti-LT-ß-R monoclonal antibody is CBE11.

51. The pharmaceutical composition according to claim 47, wherein at least two LT-ß-R activating agents comprise anti-LT-ß-R monoclonal antibodies which are directed against non-overlapping epitopes of LT-ß-R.

52. The pharmaceutical composition according to claim 51, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1 and BDA8, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of BCG6, BHA10, BKA11, CDH10 and CBE11.

53. The pharmaceutical composition according to claim 51, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of BCG6 and BHA10, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1, BDA8, BKA11, CDH10 and CBE11.

54. The pharmaceutical composition according to claim 51, wherein one anti-LT-ß-R monoclonal antibody is selected from the group consisting of BKA11 and CDH10, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1 and BDA8, BCG6, BHA10, and CBE11.

55. The pharmaceutical composition according to claim 51, wherein one anti-LT-ß-R monoclonal antibody is CBE11, and another anti-LT-ß-R monoclonal antibody is selected from the group consisting of AGH1, BDA8, BCG6, BHA10, BKA11, CDH10 and CBE11.

56. The pharmaceutical composition according to claim 51, wherein the anti-LT-ß-R monoclonal antibodies are CBE11 and BHA10.

57. The pharmaceutical composition according to claim 51, wherein the anti-LT-ß-R monoclonal antibodies are CBE11 and CDH10.

58. The pharmaceutical composition according to claim 51, wherein the anti-LT-ß-R monoclonal antibodies are AGH1 and CDH10.

59. The pharmaceutical composition according to any one of claims 51-58, further comprising IFN-γ as one of the LT-ß-R activating agents.

60. A pharmaceutical composition comprising a therapeutically effective amount of cross-linked anti-LT-ß-R antibodies as a LT-ß-R activating agent and a pharmaceutically acceptable carrier.

61. The pharmaceutical composition according to claim 60, wherein the cross-linked anti-LT-ß-R antibodies are non-covalently immobilized on a surface.

62. The pharmaceutical composition according to claim 60, wherein the cross-linked anti-LT-ß-R antibodies are covalently immobilized on a surface.

63. The pharmaceutical composition according to claim 60, wherein the cross-linked anti-LT-ß-R antibodies are non-covalently aggregated in solution by means of an anti-LT-ß-R antibody cross-linking agent.

64. The pharmaceutical composition according to claim 63, wherein the anti-LT-ß-R antibody cross-linking agent comprises a secondary antibody directed against the anti-LT-ß-R antibody.

65. The pharmaceutical composition according to claim 60, wherein the cross-linked anti-LT-ß-R antibodies are covalently aggregated in solution by means of a chemical anti-LT-ß-R antibody cross-linking agent.

66. The pharmaceutical composition according to any one of claims 60-65, further comprising IFN-γ as a second LT-ß-R activating agent.

67. A pharmaceutical composition comprising a therapeutically effective amount of at least one LT-ß-R

activating agent without exogenous LT-α/ß heteromeric complex, and a pharmaceutically acceptable carrier.

68. The pharmaceutical composition according to claim 67, wherein at least one LT-ß-R activating agent comprises an anti-LT-ß-R antibody.

69. The pharmaceutical composition according to claim 68, wherein the anti-LT-ß-R antibody is CBE11.

70. An LT-ß-R activating agent selected according to the method of claim 36.